Acute coronary syndrome

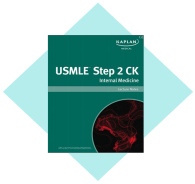
Objectives:

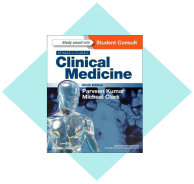
- Know the major risk factors for atherosclerosis.
- Understand the pathophysiology of atherosclerosis.
- Know the classification of acute ischemic chest pain.
- General approach to assessing an ischemic chest pain.
- General approach to the diagnostic work up of ACS.
- Know the universal definition of myocardial infarction.
- General lines of management of ACS, with emphasis on reperfusion therapy of STEMI including the indication of administering fibrinolytic therapy, and knowing the absolute contraindication for its use.
- General understanding of the immediate and late complications of STEMI.

[Color index : Important | Notes | Extra]

Resources:

- 435 slides





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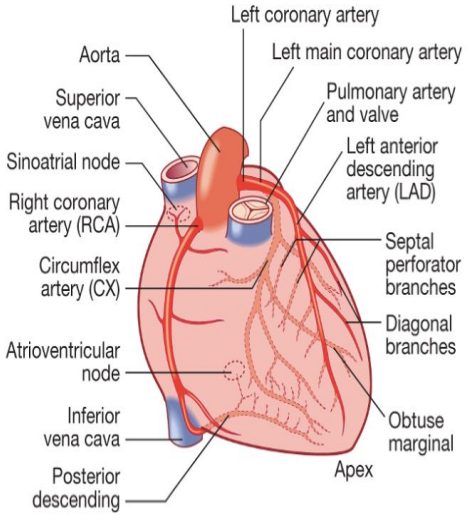
Anatomy

The coronary circulation

The left main and right coronary arteries arise from the left and right sinuses of the aortic root, distal to the aortic valve (Fig. 18.3). Within 2.5 cm of its origin, the left main coronary artery divides into the left anterior descending artery (LAD), which runs in the anterior interventricular groove, and the left circumflex artery (CX), which runs posteriorly in the atrioventricular groove. The LAD gives branches to supply the anterior part of the septum (septal perforators) and the anterior, lateral and apical walls of the LV. The CX gives marginal branches that supply the lateral, posterior and inferior segments of the LV. The right coronary artery (RCA) runs in the right atrioventricular groove, giving branches that supply the RA, RV and infero-posterior aspects of the LV. The posterior descending artery runs in the posterior interventricular groove and supplies the inferior part of the interventricular septum. The RCA supplies the sinoatrial (SA) node in about 60% of individuals and the AV node in about 90%. Proximal occlusion of the RCA therefore often results in sinus bradycardia and may also cause AV nodal block. Abrupt occlusions in the RCA, due to coronary thrombosis, result in infarction of the inferior part of the LV and often the RV. Abrupt occlusion of the LAD or CX causes infarction in the corresponding territory of the LV, and occlusion of the left main coronary artery is usually fatal.



Pathophysiology and risk factors of atherosclerosis

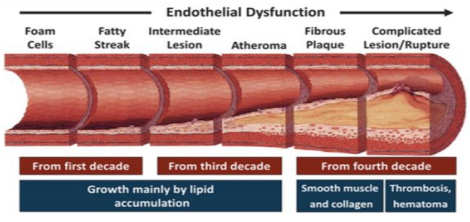
- ## Introduction:

Myocardial ischemia occurs when the oxygen demand exceeds the supply. The most common of that is obstructed coronary artery in the form of coronary atherosclerosis[It is also known as coronary artery disease (CAD)].

- ## Pathophysiology: (Endothelial cells are central to the pathogenesis of ACS)

- It's a complex inflammatory process characterized by an intimal plaque that obstructs the blood flow. It consists of : 1) Necrotic lipid core. 2) Fibromuscular cap.

1) **Lipid core formation**: It starts with endothelial damage and increased permeability that allows leakage and accumulation of oxidized lipids into the intima. Resulting in formation of flat yellow dots or lines on the endothelium called "fatty streaks". Those lipids are then taken up by the macrophages resulting in formation of "foam cells".
2) **Fibromuscular cap formation**: Cytokines are released such as Platelet-derived growth factor (PDGF) and Transforming growth factor beta (TGF-β) from the macrophages and damaged endothelial cells. Resulting in further accumulation of macrophages + migration and proliferation of smooth muscle cells. Then collagen is produced with large amounts from the smooth muscle cells.



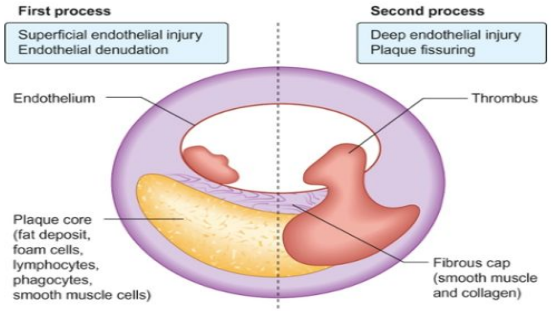
- **Unstable "complicated" plaque:**

It is formed when the plaque either ruptures superficially or with a deep endothelial fissuring.

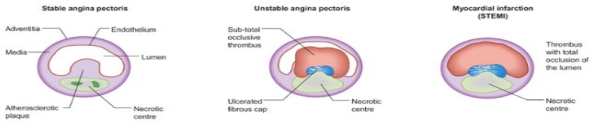
More explanation:

First process: superficial endothelial injury → exposed endothelial covering of the plaque → platelet adhesion as a result of reaction with collagen → thrombus formation on the surface of the plaque.

Second process: deep endothelial fissuring → blood enters the plaque itself → tissue factors and exposed collagen (which are highly thrombogenic) result in thrombus formation → thrombus expand into the lumen.



CAD gives rise to a wide variety of clinical presentations, ranging from relatively stable angina through to the acute coronary syndromes of unstable angina and myocardial infarction (In NSTEMI, the occlusion size is between USA and STEMI).



Risk factors:

CAD is an atherosclerotic disease that is multifactorial in origin, giving rise to the risk factor concept. Certain living habits promote atherogenic traits in genetically susceptible persons. A number of 'risk' factors are known to predispose to the condition . Some of these, such as age, gender, race and family history, cannot be changed, whereas other major risk factors, such as serum cholesterol, smoking habits, diabetes and hypertension, can be modified. Known modifiable risk factors explain >90% of the occurrence of MI in populations around the world.

Most important risk factors:		**Less reliable risk factors:**
Modifiable	**Non-modifiable**	hard to measure or determine their role in the disease
- **Diabetes mellitus:** the worst risk factor, and 1 out of 4 saudis has DM. - **Smoking.** - Hypertension. - Hyperlipidemia (**high** LDL and **low** HDL)	- Patient's age is above 45 in **men** and above 55 in **women**. - Family history of premature CAD, it should be: ➔ In **first-degree** relatives. ➔ in males under 55; females under 65. (premature).	- Physical inactivity - Poor diet - Emotional stress (indirect, it comes in association with other factors like sedentary lifestyle) - Excess alcohol ingestion

Coronary artery disease CAD (AKA : Ischemic heart disease and atherosclerotic heart disease) :

Imbalance between coronary blood supply and O_2 demand.

A. Angina :

Classical (Stable) angina	characterized by chest pain that is described as 'heavy', 'tight' or 'gripping'. Typically, the pain is central/retrosternal and may radiate to the jaw and/or arms. The pain tends to occur with exercise or emotional stress, or when walking up slopes in cold weather, and eases rapidly with rest.
Unstable angina	refers to angina of recent onset (<24 h) or deterioration in previous stable angina, with symptoms frequently occurring at rest: that is, **acute coronary syndrome.**
Refractory angina	refers to patients with severe coronary disease in whom revascularization is not possible and angina is not controlled by medical therapy.
Vasospastic or variant (Prinzmetal's) angina	refers to angina that occurs without provocation, usually at rest, as a result of coronary artery spasm. It occurs more frequently in women. Characteristically, there is **ST segment elevation on the ECG during the pain** (Like STEMI)
Microvascular angina	patients have exercise-induced angina but normal or unobstructed coronary arteries (on coronary angiography, CTCA).

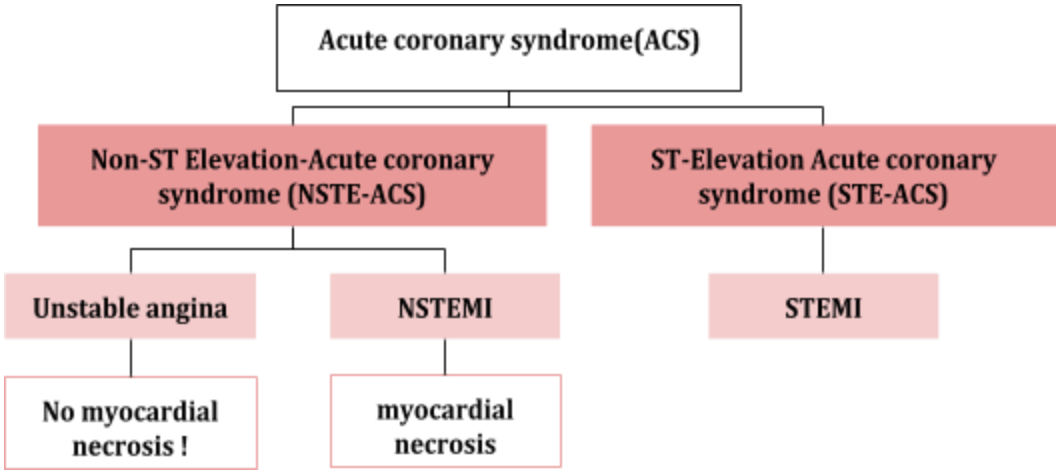
B. Acute coronary syndrome (ACS) :

It is mainly due to decreased coronary blood supply. It has an acute presentation of ischemic chest pain. Patients already have an **atherosclerotic plaque that ruptures and occludes the artery** within minutes producing the abrupt presentation. Thus, most patients have a history of stable angina.

- The third universal definition of MI:

  Typical rise in cardiac CK-MB, **troponin I or T** with at least one of the following:

  1. Ischemic symptoms.
  2. Pathological Q wave on ECG.
  3. Ischemic ECG changes e.g. ST elevation, ST depression, LBBB (left bundle branch block).
  4. Imaging evidence of new loss of viable myocardium or new WMA (wall motion abnormality).
  5. Identification of an intracoronary thrombus by angiography or autopsy.



Acute coronary syndrome (ACS)	
ST-elevation myocardial infarction (STEMI)	**Severe** ACS presentation. Usually lasts for more than 20 mins. **ECG shows ST elevation** due to transmural infarction, which is an infarction that involves the whole myocardium thickness. Although it might be asymptomatic in one-third of patients, and that is typically seen in postoperative patients, elderly, diabetic, and women. **The pain does not usually respond to sublingual glyceryl trinitrate.**
non-ST-elevation myocardial infarction (NSTEMI)	Usually **more** than 20 minutes. **ECG shows ST depression** due to subendocardial infarction (tissue necrosis). Subendocardial infarction indicates that only the first third of the myocardium is infarcted, if the blood doesn't return it progresses to STEMI.
Unstable angina (UA)	Usually **less** than 20 minutes. ECG shows ST depression due to tissue ischemia. Pain is relieved by nitroglycerin. The following patients may be said to have USA: ❖ Patients with chronic angina with increasing frequency, duration, or intensity of chest pain. ❖ Patients with new-onset angina that is severe and worsening. ❖ patients with angina at rest.

What are the differences between USA and NSTEMI? Unstable angina is a tissue ischemia; NSTEMI is a tissue necrosis.Also, in NSTEMI cardiac enzymes levels are elevated, while in USA cardiac enzymes levels are are normal WHY? Because cells are not dead (no necrosis).

When a patient comes to you with chest pain and you are suspecting ACS,the first thing you should ask for is ECG. Based on ECG you classify the condition either ST-Elevation or Non-ST-Elevation. So, it is all based on the ECG but further investigations should be done. The next step is to investigate his enzymes.

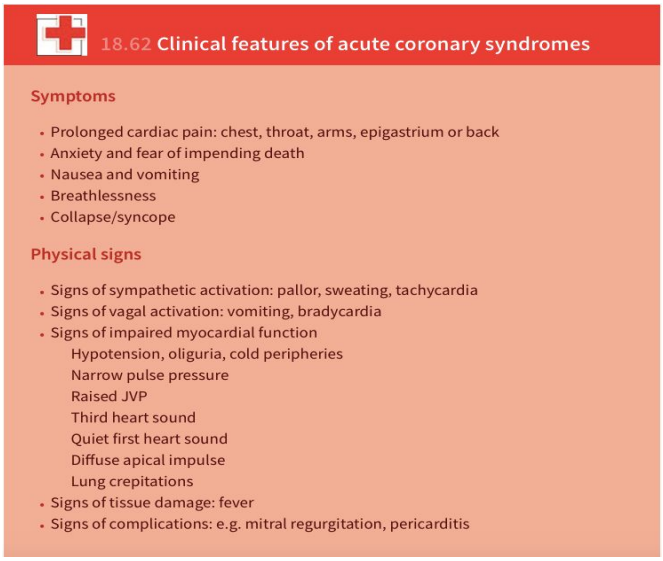
- Clinical features: (See Talley page 46)

Patients commonly present with new onset ischemic chest pain. Physical examination is usually normal, but it is done to determine any precipitating causes of myocardial infarction (eg. aortic dissection). Remember asking about risk factors i s very important in the diagnosis.

Ischemic pain Characteristics:

- **Substernal pain** that may radiate to shoulders, arms, and jaws.
- Character: **dull, heavy**, sore, **pressure-like**, squeezing pain.
- It's usually not ischemic if it's pleuritic (changes with respiration), positional (changes with position) or tender to touch.
- If the pain lasts for only a few seconds, it's usually not ischemic.
- If the pain is sharp (knifelike or pointlike) **it's NOT ischemic !**

➔ **Nonspecific associated symptoms:** (Dyspnea, Diaphoresis, Weakness and Nausea).



18.62 Clinical features of acute coronary syndromes

Symptoms

- Prolonged cardiac pain: chest, throat, arms, epigastrium or back
- Anxiety and fear of impending death
- Nausea and vomiting
- Breathlessness
- Collapse/syncope

Physical signs

- Signs of sympathetic activation: pallor, sweating, tachycardia
- Signs of vagal activation: vomiting, bradycardia
- Signs of impaired myocardial function
  - Hypotension, oliguria, cold peripheries
  - Narrow pulse pressure
  - Raised JVP
  - Third heart sound
  - Quiet first heart sound
  - Diffuse apical impulse
  - Lung crepitations
- Signs of tissue damage: fever
- Signs of complications: e.g. mitral regurgitation, pericarditis

- Electrocardiogram: It usually done to monitor the ST-segment.
  - **We see ST-segment depression** with **unstable angina** and **NSTEMI**. We can differentiate between them with the biochemical markers test.
  - **ST-segment elevation** is seen with **STEMI** due to transmural ischemia. ST segment elevation must be **higher than 1 mm** and seen in at least two leads. Otherwise, it's not considered elevation.
  - **A T-wave inversion and Q wave** (not present normally) are **highly suggestive of ACS**. They may not appear during the first day of onset, so your diagnosis can't be based upon them.
  - **Note that normal ECG does not exclude the possibility of ACS.**
  - ECG must be performed as soon as the patient presents to the ER. In fact, **ECG must be performed** upon anyone who presents with chest pain of any cause.

Typical ECG changes in myocardial infarction (STEMI)

Infarct site	Leads showing ST elevation
Anterior	
Small	V_3–V_4
Extensive	V_2–V_5
Anteroseptal	V_1–V_3
Anterolateral	V_4–V_6, I, AVL
Lateral	I, AVL
Inferior	II, III, AVF
Posterior	V_1, V_2 (reciprocal)
Sub-endocardial	Any lead
Right ventricle	VR_4

ECG changes are usually confined to the ECG leads that 'face' the infarction. The presence of new ST elevation (due to opening of the K^+ channels) of ≥0.2 mV at the J-point in leads V_1–V_3 and ≥0.1 mV in other leads, suggests anterior MI. An inferior wall MI is diagnosed when ST elevation is seen in leads II, III and AVF . Lateral MI produces changes in leads I, AVL and V_5/V_6. In patients with a posterior MI, there may be ST depression in leads V_1–V_3 with a dominant R wave, and ST elevation in lead V_5/V_6.

- ## Biochemical markers (For myocardial necrosis):

  MI causes release of certain enzymes and proteins into the bloodstream.

Creatine Kinase (CK)	- Creatin Kinase (CK) is released from multiple organs such as the myocardium , skeletal muscles, and the brain. - The Iso-form **CK-MB, is cardio-specific** - **Starts to rise 4-6 hrs after onset of ischemia**, then falls within 48-72hrs.
Troponin	- **Cardio-specific proteins** - Troponin I, and T are the most sensitive & specific markers for myonecrosis. - **Released with 4-6hrs**, but can last upto 2 week

➔ Elevated **CK-MB** and **troponin-I** indicate STEMI or NSTEMI.

➔ Normal CK-MB and troponin-I indicates unstable Angina.

➔ Remember that CK-MB is used to detect reinfarction; troponin-I is the most sensitive and specific marker.

You'll find **similar ECG findings in both NSTEMI and Unstable angina**, so we need biochemical markers to differentiate between the two:

Markers	Elevated	Normal	Indication
Troponin-I & T + CK-MB	**+++**	-	STEMI or NSTEMI
Troponin-I & T + CK-MB	-	**+++**	Unstable Angina

- ## Stress testing: Exercise tolerance test (ETT) is a tool used to evaluate chest pain when the etiology is not clear and ECG is not diagnostic. The idea here is that we're increasing the oxygen consumption of the heart.

Stress ECG:	ECG is recorded before, during and after exercise on a treadmill. It's very sensitive when the patient has normal ECG at rest. Findings aren't going to be different. So the result will be ST-segment depression due to subendocardial ischemia (Unstable angina, stable angina). Don't perform the test if ACS is not yet stabilized.
Stress Echocardiogram:	performed before and immediately after exercise. It detects wall motion abnormalities (dyskinesis, akinesis or hypokinesis) that are not present at rest. It is also used when we cannot read the ECG due to a baseline abnormalities. It is more sensitive than stress ECG in detecting ischemia.
Stress nuclear isotopes uptake:	we use thallium or sestamibi. If the myocardium is not affected and alive it will pick up the nuclear isotope. Decreased uptake will indicate ischemia.

Pharmacological stress test:	In some cases the patient can't perform the ETT so we use pharmacological stress test. The idea here is that we use drugs that increases myocardial oxygen consumption. We use that with any of the tests mentioned above (nuclear isotopes, Echocardiogram or ECG).

Use of Exercise Tolerance Testing

Test	Exercise tolerance	Exercise thallium	Exercise echo	Dipyridamole thallium	Dobutamine echo
Indication	Determine presence of ischemia	Inability to read the EKG, baseline ST segment abnormalities	Same as exercise thallium	Inability to exercise to target heart rate	Same as dipyridamole thallium
Ischemia detected	ST segment depression	Decreased uptake of nuclear isotope	Wall motion abnormalities	Decreased uptake of nuclear isotope	Wall motion abnormalities

*This schedule summarizes the stress tests, indications and ischemia detected.

complications of ACS

- Congestive heart failure:

Most common cause of in-hospital mortality. If severe may lead to cardiogenic shock (insufficient cardiac output).

- Arrhythmias[1]:

Includes:	Notes
Atrial fibrillation - Ventricular fibrillation	• (immediately use defibrillator and CPR).
Ventricular tachycardia	• **Most common cause of death in first few days after MI** is ventricular arrhythmia either VT or V-Fib
Sinus tachycardia	• May be caused by pain, anxiety or fever and it worsens ischemia
Sinus bradycardia	• Commonly occurs during early stages of acute MI, especially right-sided MI. • It might be a protective mechanism[2].
Asystole and **AV block**	• associated with ischemia involving conduction tracts). It usually appears within first 24 hours; and as time pasts it becomes less likely to happen.

[1] Will be discussed in an upcoming lecture
[2] By reducing Oxygen demand.

- ## Recurrent infarction:

Usually diagnosed with heart enzymes (remember which enzyme?). Also ST-segment elevation within the first 24 hours after the first infarction may indicate reinfarction.

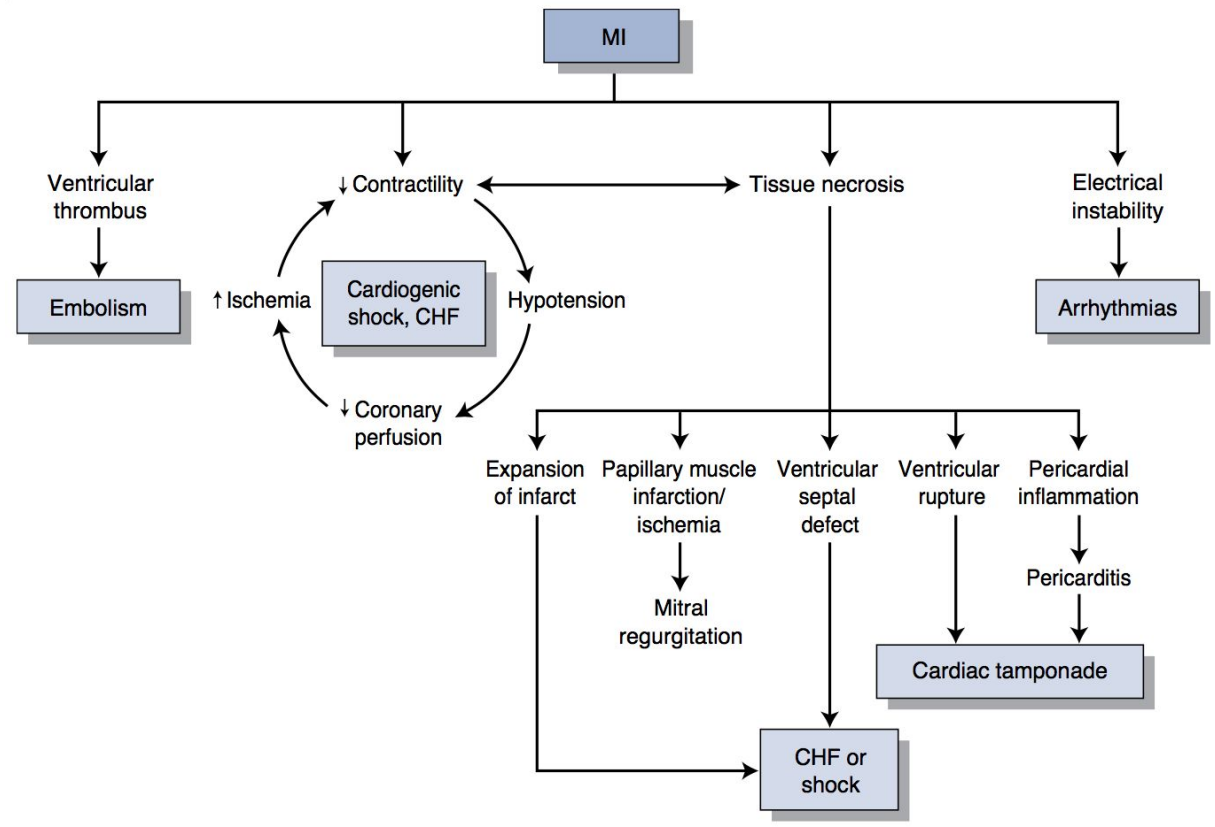
- ## Mechanical complications:

- **Free wall rupture**: leads to cardiac tamponade. Has a high mortality rate (90%).
- **Rupture of interventricular septum**: has a better chance with therapy than free wall rupture.
- **Papillary muscle rupture**: leads to mitral insufficiency and mitral regurgitation.
- **Ventricular aneurysm**: rarely ruptures, associated with tachyarrhythmia.
- **Ventricular pseudoaneurysm**: incomplete free wall rupture (pericardium is maintained), unlike the ventricular aneurysm, it tends to rupture.

- ## Dressler syndrome:

Transmural infarction with an inflammation within the pericardium. It'll then expose the pericardial antigens to the immune system resulting in formation of antibodies against the pericardium. Which will then result in autoimmune pericarditis.

Consists of: fever, malaise, pleuritis, leukocytosis in addition to pericarditis. It usually occurs after weeks or months from an MI.



*It is important to notice that some of them may lead to another.

- **The aim of the therapy is to:**

➔ **Open Artery and Improve oxygen supply :**

1. Supplemental O_2
2. Coronary vasodilators (Nitroglycerine)
3. Antiplatelet agents
4. Reperfusion therapy (Fibrinolytic therapy / Primary Percutaneous coronary intervention (PCI))
5. Antithrombotic agents.

➔ **Reduce O2 demand :**

1. Beta blockers (Propranolol, Metoprolol)
2. Analgesics (Morphine)

➔ **Other medications :**

- ACE inhibitors(Enalapril, Lisinopril).
- Statin therapy.

Acute medical management medical treatment:

Aspirin (A MUST)	Antiplatelet. Initial therapy.
Clopidogrel[3]	Antiplatelet. In patients with aspirin allergy. and if combined with aspirin they may show greater effect in reducing mortality, as it has additive effect to aspirin.
Heparin (IV)	Antithrombotic (anticoagulant). In case of STEMI, give it after the thrombolytics/PCI. Studies show that LMWH is superior to unfractionated heparin. Prevents further thrombosis and aids in insuring patency of the occluded artery.
β -blockers[4]	Not time dependent; can be given at any time during the admission.
Statins	Lipid controlling agent. Give them to everyone, but they're especially beneficial in patient with LDL> 100 mg/dL
ACE inhibitors	Give them to everyone, but they're especially beneficial in patient with ejection fraction< 40% (heart failure; often not a long-term HF)
Nitrates e.g.nitroglycerin[5]	Dilate coronary arteries (increase supply); dilates systemic veins (decrease preload and thus O_2 demand). No clear mortality benefit, but they improve symptoms. Not helpful alone
Oxygen	No clear mortality benefit, but they improve symptoms.
Morphine	Analgesics and venodilators. No clear mortality benefit, but they improve symptoms.
Calcium channel blocker (CCBs)	Used when patient can't tolerate β -blockers e.g. asthmatic patients or patients with cocaine-induced chest conditions.

[3] Blocks ADP($P2Y_{12}$), which prevent the expression of IIb/IIIa on platelets surface. Thus, inhibits platelet aggregation.(antiplatelet). Ticagrelor and prasugrel have same MOA and may be used instead in some cases.

[4] **Contraindicated** in asthmatic, diabetic and hypertensive patients

[5] It's also taken by stable angina patients. It can be taken through many routes, but the most famous two are oral and sublingual. If sublingual it'd have fast onset of action, so they take it once the attack starts or if they are about to exercise. If they take it orally, they'd take it in a regular pattern.

Acute reperfusion therapy: Reperfusion therapy is the ultimate destination,you have to open the artery either chemically(Thrombolytics) or mechanically(PCI).

1- Fibrinolytics (medical): For **STEMI ONLY,** Prompt reperfusion therapy (door to needle time <30 min) will reduce the death rate following MI.

- ONLY USED FOR STEMI (NOT NSTEMI)
- Reduces short and long term mortality
- **Should be given during a 12hr window**, and given ASAP. There is no benefit if you give it after 12 hrs.
- If Fibrinolytics fails after 30-60 minutes, refer to PCI.[6]

There're 2 types of fibrinolytics:

1. Non Fibrin specific: (Streptokinase) we don't use it anymore
2. Fibrin specific: **Alteplase (Best choice)** - Tenecteplase(Most specific) - Reteplase

Absolute Contraindications to thrombolytic therapy	**Relative Contraindications to thrombolytic therapy**
➔ Haemorrhagic stroke or stroke of unknown origin at any time ➔ Ischaemic stroke in preceding 3 months ➔ Central nervous system damage/neoplasm/vascular malformation ➔ **Recent major trauma**/surgery/head injury (within preceding 3 weeks) ➔ Gastrointestinal bleeding within the last month ➔ Known bleeding disorder ➔ Aortic dissection	➔ Oral anticoagulant therapy ➔ Pregnancy or within 1 week postpartum ➔ Non-compressible vascular punctures ➔ Traumatic resuscitation ➔ Refractory hypertension (systolic blood pressure >180 mmHg) ➔ Advanced liver disease ➔ Internal bleeding, e.g. active peptic ulcer ➔ Dementia

The worst bleeding ever is the intracranial bleeding, so DON'T GIVE Fibrinolytics if there is a risk of intracranial bleeding.

[6] Remember Time is a muscle!

<table>
<tr><td colspan="2">2-Revascularization (surgical): An angiography must be done first. It's either via CABG or PCI</td></tr>
<tr><td>CABG (Coronary Artery Bypass Graft)[7]

2 : 25 minutes</td><td>Used when the patient has:
- Three-vessels occlusion.
- Left main coronary artery occlusion.
- Left ventricular dysfunction.</td></tr>
<tr><td rowspan="3">PCI (Percutaneous coronary intervention)

1 : 42 minutes</td><td>The procedure only aims to remove the clot, but a stent[8] could be placed in the artery to improve the outcome. Preferred treatment for STEMI, as long as it's performed within 90 minutes from patient's admission. (door to balloon time <90 minutes).</td></tr>
<tr><td>Complications of PCI:
- Rupture of coronary artery on inflation.
- Restenosis.
- Hematoma at the site of entry (e.g. femoral area hematoma).</td></tr>
<tr><td>Used when the patient has:
- One-vessel occlusion.
- Two-vessels occlusion.
- No improvement despite maximal medical therapy of ACS.</td></tr>
</table>

PCI or thrombolytics?

USA or NSTEMI: PCI (we don't usually consider thrombolytic therapy for these two)

NSTEMI: PCI generally is preferred if performed by a skilled physician and within 90 minutes. It's important to note that it's only performed when reversible signs of MI are present; if the patient's cells are already dead there would be no point in revascularization surgery.

[7] Requires open heart surgery.

[8] Either a bare metal stent, or a drug-eluting stent. The latter is better.

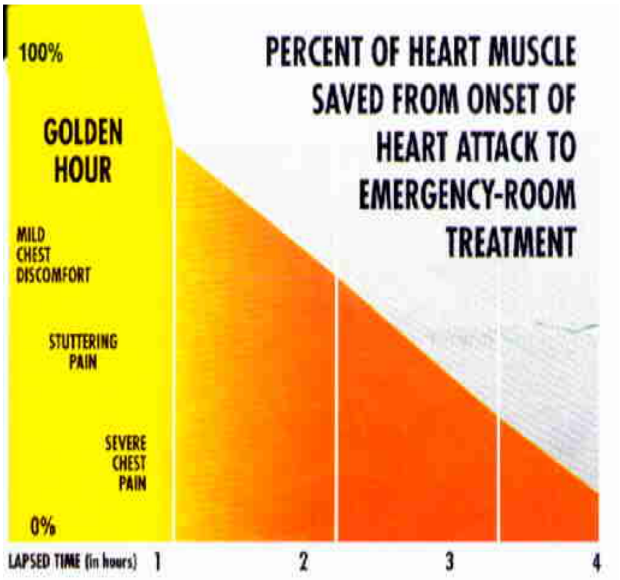
Post-discharge treatment:

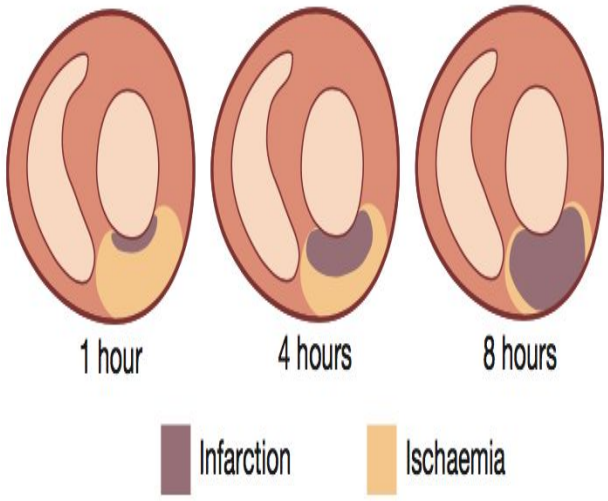
- **Aspirin**.
- β -blockers.
- Statins (especially if LDL level is more than 100 mg/dL).
- ACE inhibitors. (very important if the ejection fraction is decreased)

Mnemonic (discharge medications after ACS) : ABCDE
A: Aspirin and anti-anginals
B : Beta blockers and blood pressure
C : Cholesterol and cigarettes
D : Diet and diabetes
E : Education and exercise

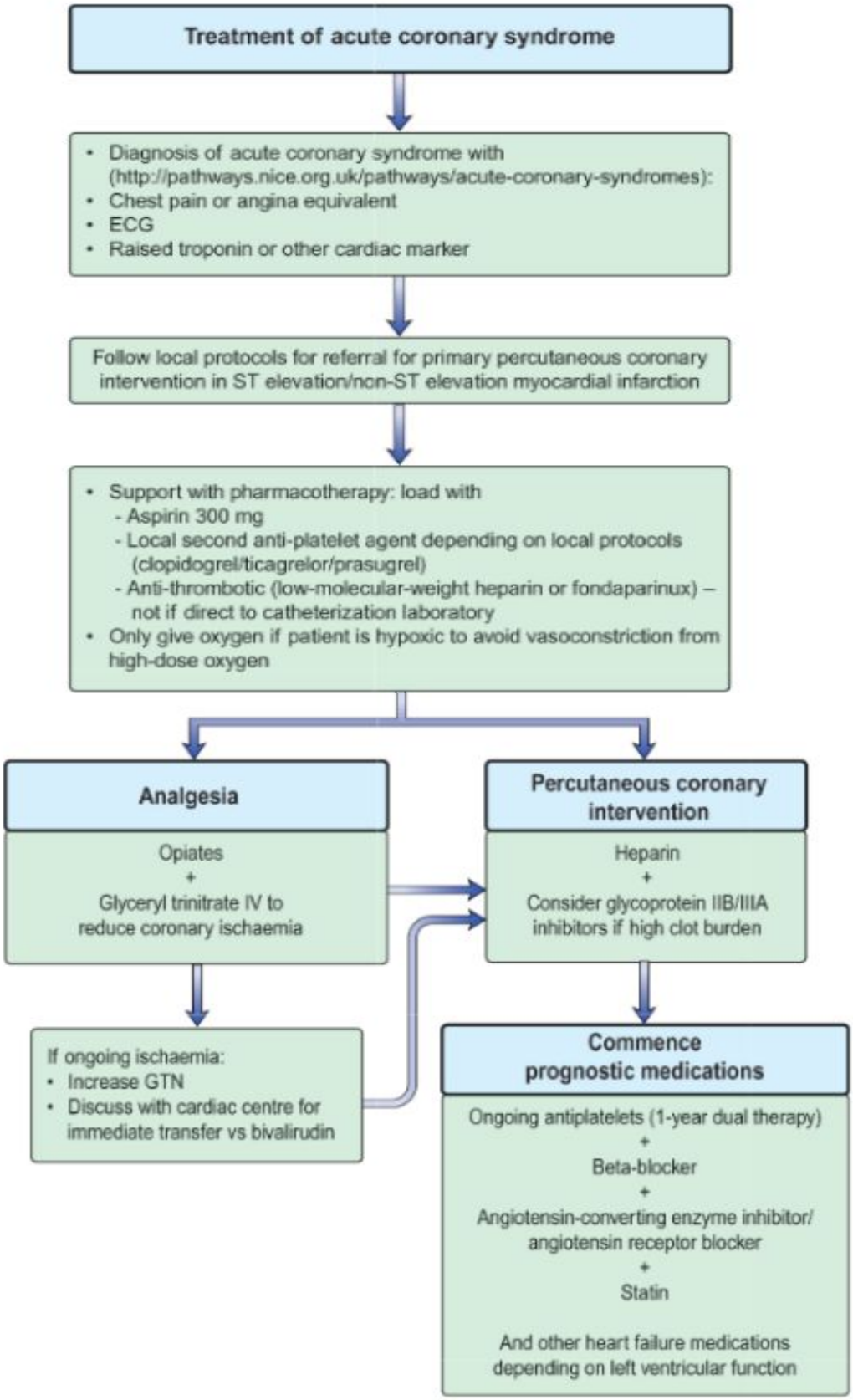
Risk factors modification:

- Try to modify the risk factors mentioned above.
- Smoking cessation among other risk factors modification shows the most immediate effect.
- The goal of LDL level is below 100 mg/dL. Statins ,among other lipid lowering agents, is the only one that reduces mortality rate.





Time is important! As we move forward damage progresses and infarction size increases; it gets harder to manage the patient and more serious complications probability increases. When it's been already 12 hours since the onset of pain there would be no point in the acute management as the injury has already been irreversible (TIME IS MUSCLE).


Treatment of acute coronary syndrome
• Diagnosis of acute coronary syndrome with (http://pathways.nice.org.uk/pathways/acute-coronary-syndromes):
• Chest pain or angina equivalent
• ECG
• Raised troponin or other cardiac marker
Follow local protocols for referral for primary percutaneous coronary intervention in ST elevation/non-ST elevation myocardial infarction
• Support with pharmacotherapy: load with
- Aspirin 300 mg
- Local second anti-platelet agent depending on local protocols (clopidogrel/ticagrelor/prasugrel)
- Anti-thrombotic (low-molecular-weight heparin or fondaparinux) – not if direct to catheterization laboratory
• Only give oxygen if patient is hypoxic to avoid vasoconstriction from high-dose oxygen
Analgesia
Opiates
+
Glyceryl trinitrate IV to reduce coronary ischaemia
Percutaneous coronary intervention
Heparin
+
Consider glycoprotein IIB/IIIA inhibitors if high clot burden
If ongoing ischaemia:
• Increase GTN
• Discuss with cardiac centre for immediate transfer vs bivalirudin
Commence prognostic medications
Ongoing antiplatelets (1-year dual therapy)
+
Beta-blocker
+
Angiotensin-converting enzyme inhibitor/ angiotensin receptor blocker
+
Statin
And other heart failure medications depending on left ventricular function

1- A 55-year-old man has just arrived in accident and emergency complaining of 20 minutes of central crushing chest pain. Which feature is most indicative of myocardial infarction at this moment in time?

A. Inverted T waves
B. ST depression
C. ST elevation
D. Q waves
E. Raised troponin

2- A 60-year-old man presents to accident and emergency with a 3-day history of increasingly severe chest pain. The patient describes the pain as a sharp, tearing pain starting in the centre of his chest and radiating straight through to his back between his shoulder blades. The patient looks in pain but there is no pallor, heart rate is 95, respiratory rate is 20, temperature 37°C and blood pressure is 155/95 mmHg. The most likely diagnosis is:

A. Myocardial infarction
B. Myocardial ischaemia
C. Aortic dissection
D. Pulmonary embolism
E. Pneumonia

3- A 49-year-old man is rushed to accident and emergency complaining of a 20-minute history of severe, crushing chest pain. After giving the patient glyceryl trinitrate (GTN) spray, he is able to tell you he suffers from hypertension and type 2 diabetes and is allergic to aspirin. The most appropriate management is:

A. Aspirin
B. Morphine
C. Heparin
D. Clopidogrel
E. Warfarin

4- You have been asked to evaluate a 42-year-old white male smoker who presented to the emergency department with sudden onset of crushing substernal chest pain, nausea, diaphoresis, and shortness of breath. His initial ECG revealed ST segment elevation in the anteroseptal leads. Cardiac enzymes were normal. The patient underwent emergent cardiac catheterization, which revealed only a 25% stenosis of the left anterior descending (LAD) artery. No percutaneous intervention was performed. Which of the following interventions would most likely reduce his risk of similar episodes in the future?

A. Placement of a percutaneous drug-eluting coronary artery stent
B. Placement of a percutaneous non–drug-eluting coronary artery stent
C. Beginning therapy with an ACE inhibitor
D. Beginning therapy with a beta-blocker

E. Beginning therapy with a calcium-channel blocker

5 - A 44-year-old woman attends her local accident and emergency department with a history of at least six months of frequent central chest pain in the early morning or during the night. She had no chest pain on exertion. This had been a particularly severe attack, lasting over 2 hours. Her pulse rate is 84/minute in sinus rhythm, and blood pressure is 134/86 mmHg. The ECG shows anterior ST segment elevation, but troponin levels do not rise. Subsequent coronary angiography is normal. What is the most likely diagnosis?

A. MI
B. Stable angina
C. Unstable angina
D. Anxiety
E. Variant Prinzmetal angina

6- A 56-year-old man presents to the accident and emergency department with a 2-hour history of central chest pain radiating to the left arm. He is anxious, nauseated and sweaty. His pulse rate is 120/minute in sinus rhythm and the ECG reveals ST elevation in leads II, III and aVF. The troponin level is significantly raised. This is certainly acute MI. Which is the most likely coronary vessel to be occluded?

A. Circumflex artery
B. Left anterior descending artery
C. Right coronary artery
D. Left main coronary artery
E. Posterior descending artery

7- A 58-year-old man has made an excellent functional recovery after an anterior MI. He is entirely asymptomatic and there is no abnormality on physical examination. His blood pressure is 134/78 mmHg and he is undertaking a cardiac rehabilitation programme. Which of the following would you <u>not</u> recommend as part of his secondary prevention planning?

A. Aspirin
B. Lisinopril
C. Simvastatin
D. Bisoprolol
E. Omega-3 fatty acids

8- A 56-year-old man is admitted to the hospital for chest pain of 2-hour duration. His heart rate is 42 bpm, with sinus bradycardia on ECG, as well as ST-segment elevation in leads II, III, and aVF. Which of the following is the most likely diagnosis?

A. He is likely in good physical condition with increased vagal tone.
B. He likely has suffered an inferior wall MI.
C. He likely has an LV aneurysm

D. The low heart rate is a reflection of a good cardiac ejection fraction.

9- A 59-year-old diabetic woman had suffered an acute anterior wall MI. Five days later, she gets into an argument with her husband and complains of chest pain. Her initial ECG shows no ischemic changes, but serum cardiac troponin I levels are drawn and return mildly elevated at this time. Which of the following is the best next step?

A. Thrombolytic therapy.
B. Percutaneous coronary intervention.
C. Coronary artery bypass.
D. Perform serial ECGs and obtain CK-MB.
E. Prepare the patient for dialysis.

10- A 59-year-old male smoker complains of severe substernal squeezing chest pain of 30-minute duration. The paramedics have given sublingual nitroglycerin and oxygen by nasal cannula. His blood pressure is 110/70 mmHg and heart rate 90 bpm on arrival to the emergency room. The ECG is normal. Which of the following is the best next step?

A. Echocardiography
B. Thallium stress test
C. Aspirin
D. Coronary angiography
E. Coronary artery bypass

1	2	3	4	5	6	7	8	9	10
C	C	D	E	E	C	E	B	D	C

1- C. Acute coronary syndrome is a spectrum of cardiac ischaemia-infarction determined by the presence of two out of three factors: chest pain, ECG changes and cardiac enzyme rise. Depending on these results, patients will fall into one of the following categories: unstable angina, NSTEMI or STEMI. Inverted T waves (A) and ST depression (B) are signs of ischaemia. ST elevation, Q waves and raised troponin are indicative of infarction. Initially, 'ST elevation' or 'non ST elevation' ECG changes are used to stratify each patient's risk as the results of blood tests for troponin levels (E) (which should be carried out 12 hours after the pain started) are not known and Q waves have not had time to develop. ST elevation (C) is a very good predictor of imminent infarction (positive troponin). However, if this patient is treated quickly enough with thrombolysis or primary PCI, infarction can be avoided. A patient with STEMI who goes on to have negative troponin is termed to have had an 'aborted MI'. Q waves (D) (indicating full-thickness MI) take time to develop, so 'Q wave' or 'non Q wave' MI is a diagnosis given on discharge.

2- C. All of the answer options can present as central chest pain, however the patient describes a very typical description of an aortic dissection (C), usually a severe, tearing pain that radiates toward the back though this can be to the jaw depending on the location of the dissection. An MI (A) is typically described as severe, crushing chest pain with an acute onset, this patient has been suffering from a 3-day history of chest pain which makes an infarction unlikely. Although myocardial ischaemia (B), i.e. angina, can occur for a longer period of time they tend not radiate to the back but more toward the jaw, arms or epigastrium, and again are described as crushing in nature rather than tearing. A pulmonary embolism (D) typically presents with pleuritic chest pain, cough and haemoptysis which are not present in this patient, or preceding risk factors such as long haul travel or surgery. Pneumonia (E) is associated with fever and productive coughing.

3- D. NICE guideline protocols state that in a patient with suspected MI, pain relief in the form of GTN spray or morphine should be administered. Since the patient has had an adequate response to GTN spray, further pain relief in the form of morphine (B) is unnecessary. In patients who are not allergic, 300mg of aspirin is recommended and ideally should be given in the ambulance. However, if the patient is allergic to aspirin (A) it should not be given since an anaphylactic reaction would compromise the patient's airway and does not overrule the harm from a possible MI. Although heparin (C) and warfarin (E) would provide good anticoagulant cover, they are slower to act and current guidance advises clopidogrel monotherapy (D) in those patients allergic to aspirin.

4- E. This patient's presentation and minimal coronary artery disease are most consistent with Prinzmetal variant angina. Prinzmetal angina is caused by severe spasm of an epicardial coronary artery. The area of vasospasm is often near a non-hemodynamically significant atherosclerotic lesion. Patients tend to be smokers and are often younger than patients who present with atherosclerotic coronary artery disease. In this case, the patient's mild LAD stenosis does not explain the degree of ischemia evidenced by the ST segment elevation. Percutaneous intervention has not been shown to be useful in management of Prinzmetal angina, as the culprit is transient vasospasm rather than fixed obstruction. Calcium-channel blockers are the mainstay of therapy to prevent recurrence of spasm. ACE inhibitors and beta-blockers do not prevent acute vasospasm. Of course, the patient should also be counseled to abstain from smoking.

5- E. Variant angina, sometimes called Prinzmetal's angina (E), of which this is a typical presentation. Its mechanism is controversial and even its existence has been questioned. The general view is that it is due to vasospasm in small coronary arteries and this is likely to respond to the effects of nitrates and calcium channel blockers such as verapamil. Beta-blockers are not effective and in theory could make it worse by aggravating vasoconstriction, but whether this actually happens is also controversial.

6- C. The answer is right coronary artery (C). This is the artery that supplies the inferior and posterior aspects of the left ventricle. The circumflex artery (A) would affect the anterolateral territory (leads I, aVL, V5–6). The left anterior descending artery (B) supplies the septum (leads V1–V4). The left main coronary artery (D) would include the circumflex artery and left anterior descending artery territory. The posterior descending artery (E) affects a limited portion of the posterior wall, and is associated with tall R waves in V1–2.

7- E. There is strong clinical trial evidence for the other four classes of drugs (A–D), although it is not clear how long the duration of therapy should be in each case. This benefit is applicable to normotensive patients with 'normal' LDL levels, although what constitutes normal in this case is controversial. Targets are likely to be reduced in the near future. One clinical trial did appear to shown additional benefit for the omega-3 fatty acids (E) but this was in a population where few were receiving statins. Subsequent data have not supported their routine use.

8- B. Sinus bradycardia is often seen with inferior wall MI, because the right coronary artery supplies the inferior wall of the left ventricle and the sinoatrial node. The ischemic changes in leads II, III, and aVF are in the region of the inferior leads. Understanding which leads reflect which portion of the heart allows for an understanding of the aspect of the heart that is affected. Also understanding the area of the heart perfused by the various coronary arteries allows for correlation of associated symptoms or therapy.

9- D. Diabetic patients can have myocardial ischemia or infarction with atypical or absent symptoms. Clinical suspicion is required, and a liberal use of cardiac enzymes. Troponin levels often remain elevated for 7 to 10 days and should not be used to diagnose reinfarction, especially if the levels are trending downward. New ECG findings or rapidly rising markers such as serum myoglobin or CK-MB can be used in this setting.

10- C. Aspirin is the first agent that should be used after oxygen and nitroglycerin. Aspirin use decreases mortality in the face of an acute coronary event. Because initial ECGs and cardiac enzymes may be normal in acute MI, serial studies are needed. Clinical assessment to exclude other causes of chest pain should be undertaken.